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NEWS	4	JAN 27	A new search aid, the Company Name Thesaurus, available in CA/CAPLUS
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NEWS	18	May 12	EXTEND option available in structure searching
NEWS	19	May 12	Polymer links for the POLYLINK command completed in REGISTRY
NEWS	20	May 17	FRFULL now available on STN
NEWS	21	May 27	STN User Update to be held June 7 and June 8 at the SLA 2004 Conference
NEWS	22	May 27	New UPM (Update Code Maximum) field for more efficient patent SDIs in CAPLUS
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NEWS	24	May 27	Explore APOLLIT with free connect time in June 2004
NEWS EXPRESS			MARCH 31 CURRENT WINDOWS VERSION IS V7.00A, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
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L1 386 "INOUE KAZUhide"/AU

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E11	2	TSUDA MASAHARU/AU
E12	2	TSUDA MASAhide/AU

=> s e5

L2 231 "TSUDA MAKOTO"/AU

=> s l1 and l2

L3 68 L1 AND L2

=> dup rem l3

PROCESSING COMPLETED FOR L3

L4 48 DUP REM L3 (20 DUPLICATES REMOVED)

=> s l4 and receptor

L5 28 L4 AND RECEPTOR

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L5 ANSWER 1 OF 28 MEDLINE on STN

ACCESSION NUMBER: 2004256602 IN-PROCESS

DOCUMENT NUMBER: PubMed ID: 14967069

TITLE: Ca2+ waves in keratinocytes are transmitted to sensory neurons: the involvement of extracellular ATP and P2Y2 **receptor** activation.

AUTHOR: Koizumi Schuichi; Fujishita Kayoko; Inoue Kaori; Shigemoto-Mogami Yukari; **Tsuda Makoto; Inoue Kazuhide**

CORPORATE SOURCE: Division of Pharmacology, National Institute of Health Sciences, 1-18-1 Kamiyoga, Setagaya, Tokyo 158-8501, Japan.

SOURCE: Biochemical journal, (2004 Jun 1) 380 (Pt 2) 329-38.
Journal code: 2984726R. ISSN: 1470-8728.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 20040525

Last Updated on STN: 20040527

AB ATP acts as an intercellular messenger in a variety of cells. In the present study, we have characterized the propagation of Ca2+ waves mediated by extracellular ATP in cultured NHEKs (normal human epidermal keratinocytes) that were co-cultured with mouse DRG (dorsal root ganglion)

neurons. Pharmacological characterization showed that NHEKs express functional metabotropic P2Y2 receptors. When a cell was gently stimulated with a glass pipette, an increase in [Ca2+]i (intracellular Ca2+ concentration) was observed, followed by the induction of propagating Ca2+ waves in neighbouring cells in an extracellular ATP-dependent manner. Using an ATP-imaging technique, the release and diffusion of ATP in NHEKs were confirmed. DRG neurons are known to terminate in the basal layer of keratinocytes. In a co-culture of NHEKs and DRG neurons, mechanical-stimulation-evoked Ca2+ waves in NHEKs caused an increase in [Ca2+]i in the adjacent DRG neurons, which was also dependent on extracellular ATP and the activation of P2Y2 receptors. Taken together, extracellular ATP is a dominant messenger that forms intercellular Ca2+ waves in NHEKs. In addition, Ca2+ waves in NHEKs could cause an increase in [Ca2+]i in DRG neurons, suggesting a dynamic cross-talk between skin and sensory neurons mediated by extracellular ATP.

L5 ANSWER 2 OF 28 MEDLINE on STN
 ACCESSION NUMBER: 2004089056 IN-PROCESS
 DOCUMENT NUMBER: PubMed ID: 14978347
 TITLE: ATP- and adenosine-mediated signaling in the central nervous system: chronic pain and microglia: involvement of the ATP receptor P2X4.
 AUTHOR: Inoue Kazuhide; Tsuda Makoto; Koizumi Schuichi
 CORPORATE SOURCE: Division of Biosignaling, National Institute of Health Sciences, Tokyo, Japan.. inoue@nihs.go.jp
 SOURCE: Journal of pharmacological sciences, (2004 Feb) 94 (2) 112-4.
 Journal code: 101167001. ISSN: 1347-8613.
 PUB. COUNTRY: Japan
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
 ENTRY DATE: Entered STN: 20040224
 Last Updated on STN: 20040325

AB We have been studying the role of ATP receptors in pain and already reported that activation of P2X(2/3) heteromeric channel/receptor in primary sensory neurons causes acutely tactile allodynia, one hallmark of neuropathic pain. We report here that tactile allodynia under the chronic pain state requires an activation of the P2X(4) ionotropic ATP receptor and p38 mitogen-activated protein kinase (MAPK) in spinal cord microglia. Two weeks after L5 spinal nerve injury, rats displayed a marked mechanical allodynia. In the rats, activated microglia were detected in the injured side of the dorsal horn and the level of the dually-phosphorylated active form of p38MAPK (phospho-p38MAPK) in these microglia was increased. Moreover, intraspinal administration of a p38MAPK inhibitor, SB203580, suppressed the allodynia. We also found that the expression level of P2X(4) was increased strikingly in spinal cord microglia after nerve injury and that pharmacological blockade or inhibition of the expression of P2X(4) reversed the allodynia. Taken together, our results demonstrate that activation of P2X(4) or p38MAPK in spinal cord microglia is necessary for tactile allodynia after nerve injury.

L5 ANSWER 3 OF 28 MEDLINE on STN
 ACCESSION NUMBER: 2003530524 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 14607246
 TITLE: Signaling of ATP receptors in glia-neuron interaction and pain.
 AUTHOR: Inoue Kazuhide; Koizumi Schuichi; Tsuda Makoto; Shigemoto-Mogami Yukari
 CORPORATE SOURCE: Division of Biosignaling, National Institute of Health Sciences, 1-18-1 Kamiyoga, Setagaya, 158-8501, Tokyo, Japan.. inoue@nihs.go.jp

SOURCE: Life sciences, (2003 Dec 5) 74 (2-3) 189-97.
Journal code: 0375521. ISSN: 0024-3205.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200401
ENTRY DATE: Entered STN: 20031111
Last Updated on STN: 20040106
Entered Medline: 20040105

AB ATP causes the activation of p38 or ERK1/2, mitogen activated protein kinases (MAPKs) resulting in the release of tumor necrosis factor-alpha (TNF) and Interleukin-6 (IL-6) from microglia. We examined the effect of TNF and IL-6 on the protection from PC12 cell death by serum deprivation. When PC12 cells were incubated with serum-free medium for 32 hr, their viability decreased to 30 %. IL-6 alone slightly protected the death of PC12 cells, whereas TNF alone did not show any protective effect. In the meanwhile, when PC12 cells were pretreated with TNF for 6 hr and then incubated with IL-6 under the condition of serum-free, the viability of PC12 cells dramatically increased. TNF induced an increase of IL-6 **receptor** (IL-6R) expression in PC12 cells at 4-6 hr. These data suggested that 6 hr pretreatment with TNF increased IL-6R expression in PC12 cells, leading to an enhancement of IL-6-induced neuroprotective action. To elucidate the role of p38 in pathological pain, we investigated whether p38 is activated in the spinal cord of the neuropathic pain model. In the rats displaying a marked allodynia, the level of phospho-p38 was increased in the microglia of injury side in the dorsal horn. Intraspinal administration of p38 inhibitor suppressed the allodynia. These results demonstrate that neuropathic pain hypersensitivity depends upon the activation of p38 signaling pathway in microglia in the dorsal horn following peripheral nerve injury.

L5 ANSWER 4 OF 28 MEDLINE on STN
ACCESSION NUMBER: 2003381868 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12917686
TITLE: P2X4 receptors induced in spinal microglia gate tactile allodynia after nerve injury.
COMMENT: Comment in: Nature. 2003 Aug 14;424(6950):729-30. PubMed ID: 12917663
AUTHOR: Tsuda Makoto; Shigemoto-Mogami Yukari; Koizumi Schuichi; Mizokoshi Akito; Kohsaka Shinichi; Salter Michael W; Inoue Kazuhide
CORPORATE SOURCE: Division of Biosignaling, National Institute of Health Sciences, 1-18-1 Kamiyoga, Setagaya, Tokyo 158-8501, Japan.
SOURCE: Nature, (2003 Aug 14) 424 (6950) 778-83.
Journal code: 0410462. ISSN: 1476-4687.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 20030815
Last Updated on STN: 20030822
Entered Medline: 20030821

AB Pain after nerve damage is an expression of pathological operation of the nervous system, one hallmark of which is tactile allodynia-pain hypersensitivity evoked by innocuous stimuli. Effective therapy for this pain is lacking, and the underlying mechanisms are poorly understood. Here we report that pharmacological blockade of spinal P2X4 receptors (P2X4Rs), a subtype of ionotropic ATP **receptor**, reversed tactile allodynia caused by peripheral nerve injury without affecting acute pain behaviours in naive animals. After nerve injury, P2X4R expression increased strikingly in the ipsilateral spinal cord, and P2X4Rs were induced in hyperactive microglia but not in neurons or astrocytes.

Intraspinal administration of P2X4R antisense oligodeoxynucleotide decreased the induction of P2X4Rs and suppressed tactile allodynia after nerve injury. Conversely, intraspinal administration of microglia in which P2X4Rs had been induced and stimulated, produced tactile allodynia in naive rats. Taken together, our results demonstrate that activation of P2X4Rs in hyperactive microglia is necessary for tactile allodynia after nerve injury and is sufficient to produce tactile allodynia in normal animals. Thus, blocking P2X4Rs in microglia might be a new therapeutic strategy for pain induced by nerve injury.

L5 ANSWER 5 OF 28 MEDLINE on STN
ACCESSION NUMBER: 2002323765 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12028354
TITLE: Downregulation of P2X3 **receptor**-dependent sensory functions in A/J inbred mouse strain.
AUTHOR: **Tsuda Makoto**; Shigemoto-Mogami Yukari; Ueno Shinya; Koizumi Schuichi; Ueda Hiroshi; Iwanaga Toshihiko; **Inoue Kazuhide**
CORPORATE SOURCE: Section of Neuropharmacology, Division of Pharmacology, National Institute of Health Sciences, Tokyo, Japan.
SOURCE: European journal of neuroscience, (2002 May) 15 (9) 1444-50.
Journal code: 8918110. ISSN: 0953-816X.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020618
Last Updated on STN: 20020727
Entered Medline: 20020726

AB There is large variability in the various pain responses including those to tissue injury among inbred mouse strains. However, the determinant factors for the strain-specific differences remain unknown. The P2X3 sensory-specific ATP-gated channel has been implicated as a damage-sensing molecule that evokes a pain sensation by receiving endogenous ATP from injured tissue. In this study, to clarify the contribution of the sensory P2X3 signalling to strain-specific differences in tissue injury pain, we examined whether the P2X3-mediated in vivo and in vitro responses in dorsal root ganglion (DRG) neurons are changed in the A/J inbred mouse strain, which is known to be resistant to tissue injury pain caused by formalin. Here we found that A/J mice exhibited a low magnitude of nocifensive behaviour induced by the P2X agonist alpha,beta-methylene ATP (alpha beta meATP) into the hindpaw compared with C57BL/6 J mice. This behaviour was blocked by P2X3 antisense oligodeoxynucleotides. The low magnitude of the in vivo pain sensation could be observed similarly in the in vitro response; the increase in the intracellular Ca(2+) increase by alpha beta meATP in capsaicin-sensitive DRG neurons from A/J mice was significantly lower than that from C57BL/6 J mice. In A/J DRG neurons the P2X3 protein level was significantly lower compared with C57BL/6 J DRG neurons. The change in P2X3 protein was selective because P2X2 protein was expressed equally in both strains. The present study suggests that the downregulation of sensory P2X3 could be one of the molecular predispositions to low sensitivity to tissue injury pain in the A/J inbred mouse strain.

L5 ANSWER 6 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:468825 CAPLUS
TITLE: Neuron-to-astrocyte communication by endogenous ATP in mixed culture of rat hippocampal neurons and astrocytes
AUTHOR(S): Koizumi, Schuichi; Fujishita, Kayoko; **Tsuda, Makoto**; **Inoue, Kazuhide**
CORPORATE SOURCE: Section of Neuropharmacology, Division of

Pharmacology, National Institute of Health Sciences,
Tokyo, Japan
SOURCE: Drug Development Research (2003), 59(1), 88-94
CODEN: DDREDK; ISSN: 0272-4391
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB ATP is recognized as an important intercellular signaling mol. in the peripheral and CNS. Glutamate is reported to be an important neuron-to-glia mediator being released from neurons and astrocytes that activates astrocytic and neuronal Ca^{2+} responses, resp. The authors demonstrate here that endogenous ATP could be an extracellular mol. for neuron-to-astrocyte communication in cocultured rat hippocampal neurons and astrocytes. Hippocampal neurons reveal synchronized Ca^{2+} oscillation, which was due to glutamatergic synaptic transmission. When analyzed in a fura-2 method, a slight and very slow increase in intracellular Ca^{2+} concentration ($[\text{Ca}^{2+}]_i$) elevation was observed in some population of astrocytes.

Such astrocytic $[\text{Ca}^{2+}]_i$ elevation was dramatically inhibited by apyrase, though apyrase itself had no effect on neuronal Ca^{2+} oscillation. For a detail anal., the authors investigated changes in $[\text{Ca}^{2+}]_i$ in cells using a confocal microscopy. When cocultured hippocampal neurons and astrocytes were depolarized electronically in the presence of glutamate-receptor antagonists, a transient elevation in $[\text{Ca}^{2+}]_i$ was observed in neurons, which was followed by a slowly initiated and small rise in $[\text{Ca}^{2+}]_i$ in astrocytes. Apyrase or P_2 receptor antagonists almost abolished the $[\text{Ca}^{2+}]_i$ rises in astrocytes, suggesting that depolarization-evoked ATP release from neurons should produce astrocytic $[\text{Ca}^{2+}]_i$ elevation via P_2 receptors. Using a luciferin-luciferase bioluminescence assay, the authors found that neurons could release ATP in an activity-dependent manner. Thus, endogenous ATP is an important intercellular mediator between neurons and astrocytes and functions of these cells are fine-tuned by endogenously released ATP in situ.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2003:468821 CAPLUS
DOCUMENT NUMBER: 139:208108
TITLE: ATP induced three types of pain behaviors, including allodynia
AUTHOR(S): Inoue, Kazuhide; Tsuda, Makoto; Koizumi, Schuichi
CORPORATE SOURCE: Section of Neuropharmacology, Division of Pharmacology, National Institute of Health Sciences, Tokyo, Japan
SOURCE: Drug Development Research (2003), 59(1), 56-63
CODEN: DDREDK; ISSN: 0272-4391
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB ATP excites dorsal root ganglion (DRG) neurons in the nociceptive signaling pathway via ATP-gated cation channels (P_2X receptors). ATP and its analog α,β -methylene ATP ($\alpha\beta\text{meATP}$) activated two types of inward currents; one is a rapidly desensitizing current which is observed in capsaicin (CAP)-sensitive DRG neurons and in C6BU-1 cells expressing homomeric P_2X_3 receptors. The other is a slowly desensitizing current which is seen in CAP-insensitive medium-sized DRG neurons and in C6BU-1 expressing heteromeric $\text{P}_2\text{X}_2/3$ receptors. These findings suggest that P_2X_3 and $\text{P}_2\text{X}_2/3$ are involved in the generation or modulation of pain. To clarify this hypothesis, the authors investigated the effects of agonists for P_2X receptors on pain sensitivities using a behavioral approach. Activation of P_2X receptors at a peripheral site by the injection of ATP or $\alpha\beta\text{meATP}$ into the hindpaw produced three

distinct types of pain-related behaviors (nocifensive behavior, thermal hyperalgesia, and mech. allodynia). Nocifensive behavior and thermal hyperalgesia were blocked by pretreatment with PPADS and were not observed in neonatal CAP-treated adult rats that had selectively lost CAP-sensitive neurons. The $\alpha\beta$ meATP-induced allodynia was sensitive to PPADS, was a relatively long-lasting response, and remained in neonatal CAP-treated adult rats. Furthermore, while pretreatment by P2X3 antisense oligodeoxynucleotide (ODN) diminished all three pain responses, P2X2 antisense ODN inhibited only the mech. allodynia. These findings suggests that activation of homomeric P2X3 receptors in peripheral terminals of CAP-sensitive primary afferent fibers plays a role in the induction of nocifensive behavior and thermal hyperalgesia and that activation of heteromeric P2X2/3 receptors in CAP-insensitive fibers leads to the induction of mech. allodynia.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:731084 CAPLUS

DOCUMENT NUMBER: 138:131187

TITLE: New molecules related to onset of pain. (2) ATP and non-receptor ion channels

AUTHOR(S): Inoue, Kazuhide; Tsuda, Makoto; Koizumi, Shuichi

CORPORATE SOURCE: Divison of Pharmacology, National Institute of Health Sciences, Japan

SOURCE: Opioido Chiryo (2001), 240-245. Eruzebia-Saiensu K.K. Mikusu: Tokyo, Japan. CODEN: 69DCVB; ISBN: 4-86034-614-9

DOCUMENT TYPE: Conference; General Review

LANGUAGE: Japanese

AB A review, on the roles of P2X receptors and nonreceptor voltage-dependent Na⁺-channel in nociception, discussing the distribution of P2X receptors and Na⁺ channel and the primary sensory neurons and P2X receptors- and the Na⁺-channel-mediated electrophysiol. responses in the sensory neurons and nociception.

L5 ANSWER 9 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2001:891880 CAPLUS

DOCUMENT NUMBER: 136:15563

TITLE: Mechanical allodynia caused by intraplantar injection of P2X receptor agonist in rats: involvement of heteromeric P2X2/3 receptor signaling in capsaicin-insensitive primary afferent neurons

AUTHOR(S): Tsuda, Makoto; Koizumi, Shuichi; Kita, Aya; Sigemoto, Yukari; Ueno, Shinya; Inoue, Kazuhide

CORPORATE SOURCE: Section of Neuropharmacology, Division of Pharmacology, National Institute of Health Sciences, Setagaya, Tokyo, 158-8501, Japan

SOURCE: Journal of Neuroscience (2000), 20(15), RC90/1-RC90/5 CODEN: JNRSDS; ISSN: 0270-6474

PUBLISHER: Society for Neuroscience

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Extracellular ATP has been known to activate sensory neurons via the ATP-gated ion channels P2X receptors, indicating that the P2X receptors may play a role in signal transduction of pain from the periphery to the spinal cord in vivo. Here, the authors found a novel nociceptive response induced by ATP, mech. allodynia (hypersensitivity to innocuous mech. stimulus). Injection of α,β -methylene ATP ($\alpha\beta$ meATP), an agonist to P2X receptor, into plantar surface in rats produced the mech. allodynia along with previously described nocifensive behavior and thermal hyperalgesia. This allodynic

response was blocked by pretreatment with the P2 receptor antagonist pyridoxal-phosphate-6-azophenyl-2',4'-disulfonate. Interestingly, only the mech. allodynia evoked by meATP selectively remained in neonatal capsaicin-treated adult rats that had selectively lost the capsaicin-sensitive neurons. ATP has been shown to produce two distinguishable electrophysiol. responses (inward currents with rapid and slow desensitization) in dorsal root ganglion (DRG) neurons. In the present electrophysiol. experiment, the percentage of DRG neurons that responded to meATP with slow desensitizing inward current remained constant in capsaicin-treated rats, whereas the percentage that responded with rapid desensitizing current dramatically decreased. Taken together with the authors' previous finding that the $\alpha\beta$ meATP-activated slow desensitizing current in DRG neurons is mediated by heteromeric P2X2/3 (P2X2 and P2X3) receptors, it is hypothesized that activation of heteromeric P2X2/3 receptors in peripheral terminals of capsaicin-insensitive primary afferent fibers leads to the induction of mech. allodynia.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 10 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2001:729114 CAPLUS

DOCUMENT NUMBER: 136:32081

TITLE: Mechanisms underlying extracellular ATP-evoked interleukin-6 release in mouse microglial cell line, MG-5

AUTHOR(S): Shigemoto-Mogami, Yukari; Koizumi, Schuichi; Tsuda, Makoto; Ohsawa, Keiko; Kohsaka, Shinichi; Inoue, Kazuhide

CORPORATE SOURCE: Division of Pharmacology, National Institute of Health Sciences, Tokyo, 158-8501, Japan

SOURCE: Journal of Neurochemistry (2001), 78(6), 1339-1349
CODEN: JONRA9; ISSN: 0022-3042

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Microglia play various important roles in the CNS via the synthesis of cytokines. The ATP-evoked production of interleukin-6 (IL-6) and its intracellular signals were examined using a mouse microglial cell line, MG-5. ATP, but not its metabolites, produced IL-6 in a concentration-dependent manner. Although ATP activated two mitogen-activated protein kinases, i.e. p38 and extracellular signal-regulated protein kinase, only p38 was involved in the IL-6 induction. However, the activation of p38 was not sufficient for the IL-6 induction because 2'- and 3'-O-(4-benzoylbenzoyl) ATP, an agonist to P2X7 receptors, failed to produce IL-6 despite the fact that it activated p38. Unlike in other cytokines in microglial cells, P2Y rather than P2X7 receptors seem to have a major role in the IL-6 production by the cells. The ATP-evoked IL-6 production was attenuated by Go6976, an inhibitor of Ca²⁺-dependent protein kinase C (PKC). The P2Y receptor responsible for these responses was insensitive to pertussis toxin (PTX) and was linked to phospholipase C. Taken together, ATP acting on PTX-insensitive P2Y receptors activates p38 and Ca²⁺-dependent PKC, thereby resulting in the mRNA expression and release of IL-6 in MG-5. This is a novel pathway for the induction of cytokines in microglia.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2001:487070 CAPLUS

DOCUMENT NUMBER: 136:83986

TITLE: Role of endogenous ATP at the incision area in a rat model of postoperative pain

AUTHOR(S): Tsuda, Makoto; Koizumi, Schuichi;

CORPORATE SOURCE: **Inoue, Kazuhide**
Section of Neuropharmacology, Division of
Pharmacology, National Institute of Health Sciences,
Tokyo, 158-8501, Japan
SOURCE: NeuroReport (2001), 12(8), 1701-1704
CODEN: NERPEZ; ISSN: 0959-4965
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The aim of the present study is to characterize the role of endogenous ATP leaked from damaged cells in a rat model of postoperative pain using behavioral and immunocytochem. approaches. We found that systemic (i.v.) and local (incision area) administration of a P2 **receptor** antagonist, pyridoxal-phosphate-6-azophenyl-2',4'-disulfonic acid (PPADS) before surgery significantly attenuated mech. allodynia caused by an incision of the plantar surface of the hindpaw. Furthermore, PPADS significantly reduced the incision-evoked c-Fos protein expression, a marker of neuronal activity, in the dorsal horn of the spinal cord. The present findings suggest that excitatory signaling by endogenous ATP leaked from damaged cells via PPADS-sensitive P2 receptors is necessary for the induction of the postoperative pain characterized by mech. allodynia.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2000:883971 CAPLUS
DOCUMENT NUMBER: 134:15543
TITLE: ATP receptors in pain
AUTHOR(S): **Tsuda, Makoto**; Koizumi, Schuichi;
Inoue, Kazuhide

CORPORATE SOURCE: Neuropharmacol. Sect., Div. Pharmacol., Natl. Inst.
Health Sci., 1-18-1 Kamiyoga, Setagaya, Tokyo,
158-8501, Japan

SOURCE: Nippon Yakurigaku Zasshi (2000), 116(6), 343-350
CODEN: NYKZAU; ISSN: 0015-5691

PUBLISHER: Nippon Yakuri Gakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

AB A review with 50 refs. Extracellular ATP has been known to activate sensory neurons via the ATP-gated ion channels P2X receptors, leading to the proposal that the P2X receptors may play a role in signal transduction of pain from the peripheral site to the spinal cord in vivo. P2X3 receptors are expressed in capsaicin-sensitive small-sized dorsal root ganglion (DRG) neurons, and they are involved in the generation of rapidly desensitizing inward current and evoking nocifensive behavior and thermal hyperalgesia. Heteromeric P2X2/3 (P2X2 and P2X3) **receptor** is expressed in capsaicin-insensitive primary afferent fibers, and its activation leads to the generation of slow desensitizing currents and induction of mech. allodynia. In addition, accumulating information suggests the involvement of G protein-coupled ATP receptors in the modulation of the generation and transmission of pain.

L5 ANSWER 13 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1999:810142 CAPLUS
DOCUMENT NUMBER: 132:117874
TITLE: Evidence for the involvement of spinal endogenous ATP and P2X receptors in nociceptive responses caused by formalin and capsaicin in mice
AUTHOR(S): **Tsuda, Makoto**; Ueno, Shinya; **Inoue, Kazuhide**

CORPORATE SOURCE: Section of Neuropharmacology, Division of
Pharmacology, National Institute of Health Sciences,
Tokyo, 158-8501, Japan

SOURCE: British Journal of Pharmacology (1999), 128(7),
1497-1504
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Stockton Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The aim of the present study is to characterize the role of spinal endogenous ATP and P2X receptors in the generation of neurogenic and inflammatory pain. We examined the effects of intrathecal treatment with P2X **receptor** antagonists on the formalin- and capsaicin-induced nociceptive behaviors in mice. Intrathecal pretreatment with the general P2 **receptor** antagonist, pyridoxal-phosphate-6-azophenyl-2',4'-disulfonic acid (PPADS), significantly suppressed both the first and second phases of the formalin-induced nociceptive behavior. The second phase of the nociceptive response was also suppressed by intrathecal treatment with PPADS after the first phase. Furthermore, pretreatment with the selective antagonist for the P2X1, P2X3 and P2X2+3 receptors, 2',3'-O-(2,4,6-trinitrophenyl)ATP (TNP-ATP), significantly reduced the first phase, but not the second phase. The second phase was also not suppressed by intrathecal TNP-ATP after the first phase. Capsaicin-induced nociceptive behavior that has been shown to be a model for neurogenic pain, was also significantly suppressed by intrathecal pretreatment with PPADS or TNP-ATP. Nociceptive behavior in the first phase of the formalin test and in the capsaicin test were significantly inhibited by intrathecal pretreatment with α,β -methylene ATP (α,β MeATP: 5 μ g mouse⁻¹) 15 min prior to injection of formalin or capsaicin. This treatment has been previously shown to desensitize spinal P2X3 **receptor** subtypes in vivo. These findings suggest that spinal endogenous ATP may play a role in (1) the formalin- and capsaicin-induced neurogenic pain via the PPADS- and TNP-ATP-sensitive P2X receptors which are also desensitized by α,β MeATP (perhaps the P2X3 **receptor** subtype) and (2) formalin-induced inflammatory pain via PPADS-sensitive, TNP-ATP- and α,β MeATP-insensitive P2X (and/or P2Y) receptors.

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 14 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1999:668805 CAPLUS

DOCUMENT NUMBER: 132:88221

TITLE: The functions of ATP receptors in the synaptic transmission in the hippocampus

AUTHOR(S): Inoue, Kazuhide; Koizumi, Schuichi; Ueno, Shinya; Kita, Aya; Tsuda, Makoto

CORPORATE SOURCE: Division of Pharmacology, National Institute of Health Sciences, Tokyo, 158, Japan

SOURCE: Progress in Brain Research (1999), 120(Nucleotides and Their Receptors in the Nervous System), 193-206
CODEN: PBRR44; ISSN: 0079-6123

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review with 75 refs. is presented on the possible function of ATP receptors in synaptic transmission in the hippocampus. Information was presented that ATP presynaptically inhibits the release of glutamate, an excitatory neurotransmitter from the hippocampus. Addnl., ATP may stimulate the release of GABA from some interneurons. ATP also activates microglia to release plasminogen, which promotes the development of mesencephalic dopaminergic neurons and enhances neurite outgrowth from explants of neocortical tissue. Thus, ATP may have a role in the protection of the function of neurons in the hippocampus from overstimulation by glutamate.

REFERENCE COUNT: 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 15 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1999:368929 CAPLUS

DOCUMENT NUMBER: 131:180135

TITLE: In vivo pathway of thermal hyperalgesia by intrathecal administration of α,β -methylene ATP in mouse spinal cord: involvement of the glutamate-NMDA **receptor** system

AUTHOR(S): Tsuda, Makoto; Ueno, Shinya; Inoue, Kazuhide

CORPORATE SOURCE: Section of Neuropharmacology, Division of Pharmacology, National Institute of Health Sciences, Tokyo, 158-8501, Japan

SOURCE: British Journal of Pharmacology (1999), 127(2), 449-456

CODEN: BJPCBM; ISSN: 0007-1188

PUBLISHER: Stockton Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The aim of the present study is to characterize the role of the P2X **receptor** in spinal nociceptive processing in vivo. We investigated the mechanisms of the P2X **receptor** agonist α,β -methylene ATP (α,β meATP)-induced modulation of acute nociceptive signaling in mouse spinal cord. Intrathecal administration of α,β meATP produced a significant and dose-dependent thermal hyperalgesic response. This response was completely blocked by intrathecal pretreatment with the non-selective P2 **receptor** antagonist, pyridoxal-phosphate-6-azophenyl-2',4'-disulfonate (PPADS) and the selective P2X1, P2X3 and P2X2+3 **receptor** antagonist, 2',3'-O-(2,4,6-trinitrophenyl)ATP (TNP-ATP). Pretreatment with α,β meATP 15, 30 and 60 min prior to administration of a second dose of α,β meATP diminished the α,β meATP-induced thermal hyperalgesia. A potent agonist for the P2X1 **receptor**, β,γ -methylene-L-ATP, did not show the hyperalgesic response, indicating that the P2X1 **receptor** is not involved in the spinal nociceptive pathway. In fura-2 expts. using mouse dorsal root ganglion (DRG) neurons, α,β meATP (100 μ M) increased intracellular Ca^{2+} ($[\text{Ca}^{2+}]_i$). This was not produced by a second application of α,β meATP. The same DRG neurons also showed a marked $[\text{Ca}^{2+}]_i$ increase in response to capsaicin (3 μ M). Intrathecal pretreatment with the Ca^{2+} -dependent exocytosis inhibitor, botulinum neurotoxin B, abolished the thermal hyperalgesia by α,β meATP. Furthermore, thermal hyperalgesia was significantly inhibited by the N-methyl-D-aspartate (NMDA) **receptor** antagonists, 2-amino-5-phosphonopentanoate (APV), dizocilpine and ifenprodil. These findings suggest that α,β meATP-induced thermal hyperalgesia may be mediated by the spinal P2X3 **receptor** subtype that causes unresponsiveness by repetitive agonist applications, and that α,β meATP (perhaps through P2X3 receptors) may evoke spinal glutamate release which, in turn, leads to the generation of thermal hyperalgesia via activation of NMDA receptors.

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 16 OF 28 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1999:123816 CAPLUS

DOCUMENT NUMBER: 130:279598

TITLE: Cell type-specific ATP-activated responses in rat dorsal root ganglion neurons

AUTHOR(S): Ueno, Shinya; Tsuda, Makoto; Iwanaga, Toshihiko; Inoue, Kazuhide

CORPORATE SOURCE: Department of Pharmacology, School of Medicine, Fukuoka University, Fukuoka, 814-0180, Japan

SOURCE: British Journal of Pharmacology (1999), 126(2),

429-436

CODEN: BJPCBM; ISSN: 0007-1188

PUBLISHER: Stockton Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The aim of our study is to clarify the relationship between expression pattern of P2X receptors and the cell type of male adult rat (Wistar) dorsal root ganglion (DRG) neurons. We identified the nociceptive cells of acutely dissociated DRG neurons from adult rats type using capsaicin sensitivity. Two types of ATP-activated currents, one with fast, the other with slow desensitization, were found under voltage-clamp conditions. In addition, cells with fast but not slow desensitization responded to capsaicin, indicating that there was a relationship between current kinetics and capsaicin-sensitivity. Both types of neurons were responsive to ATP and α , β methylene-ATP (α , β meATP). The concentration of α , β meATP producing half-maximal activation (EC50) of neurons with fast desensitization was less (11 μ M) than that of neurons with slow desensitization (63 μ M), while the Hill coeffs. were similar. Suramin and pyridoxal-phosphate-6-azophenyl-2',4'-disulfonic acid tetrasodium (PPADS) antagonized α , β meATP-induced currents in both types of neurons. In situ hybridization revealed that small cells of the DRG predominantly expressed mRNAs of P2X3, and medium-sized cells expressed mRNAs of P2X2 and P2X3. In contrast, both mRNAs were not detected in large cells of the DRG. These results suggest that capsaicin-sensitive, small-sized DRG neurons expressed mainly the homomeric P2X3 subunit and that capsaicin-insensitive, medium-sized DRG neurons expressed the heteromultimeric **receptor** with P2X2 and P2X3.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 17 OF 28 LIFESCI COPYRIGHT 2004 CSA on STN

ACCESSION NUMBER: 2001:80098 LIFESCI

TITLE: Mechanical Allodynia Caused by Intraplantar Injection of P2X Receptor Agonist in Rats: Involvement of Heteromeric P2X sub(2/3) Receptor Signaling in Capsaicin-Insensitive Primary Afferent Neurons

AUTHOR: Tsuda, Makoto; Koizumi, Schuichi; Kita, Aya; Shigemoto, Yukari; Ueno, Shinya; Inoue, Kazuhide

CORPORATE SOURCE: Section of Neuropharmacology, Division of Pharmacology, National Institute of Health Sciences, 1-18-1 Kamiyoga, Setagaya-ku, Tokyo 158-8501, Japan

SOURCE: Journal of Neuroscience [J. Neurosci.], (20000801) vol. 20, no. 15, pp. RC90:1-5. ISSN: 0270-6474.

DOCUMENT TYPE: Journal

FILE SEGMENT: N3

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Extracellular ATP has been known to activate sensory neurons via the ATP-gated ion channels P2X receptors, indicating that the P2X receptors may play a role in signal transduction of pain from the periphery to the spinal cord in vivo. Here, we found a novel nociceptive response induced by ATP, mechanical allodynia (hypersensitivity to innocuous mechanical stimulus). Injection of alpha, beta-methylene ATP (alpha beta meATP), an agonist to P2X **receptor**, into plantar surface in rats produced the mechanical allodynia along with previously described nocifensive behavior and thermal hyperalgesia. This allodynic response was blocked by pretreatment with the P2 **receptor** antagonist pyridoxal-phosphate-6-azophenyl-2',4'-disulfonate. Interestingly, only the mechanical allodynia evoked by alpha beta meATP selectively remained in neonatal capsaicin-treated adult rats that had selectively lost the capsaicin-sensitive neurons. ATP has been shown to produce two distinguishable electrophysiological responses (inward currents with rapid

and slow desensitization) in dorsal root ganglion (DRG) neurons. In the present electrophysiological experiment, the percentage of DRG neurons that responded to alpha beta meATP with slow desensitizing inward current remained constant in capsaicin-treated rats, whereas the percentage that responded with rapid desensitizing current dramatically decreased. Taken together with our previous finding that the alpha beta meATP-activated slow desensitizing current in DRG neurons is mediated by heteromeric P2X sub(2/3) (P2X sub(2) and P2X sub(3)) receptors, it is hypothesized that activation of heteromeric P2X sub(2/3) receptors in peripheral terminals of capsaicin-insensitive primary afferent fibers leads to the induction of mechanical allodynia.

L5 ANSWER 18 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2004:251608 BIOSIS

DOCUMENT NUMBER: PREV200400252794

TITLE: The role of ATP receptors in the pain sensation.

AUTHOR(S): Inoue, Kazuhide [Reprint Author]; Tsuda, Makoto [Reprint Author]; Koizumi, Schuichi [Reprint Author]

CORPORATE SOURCE: Div. Biosignal, NIHS, Tokyo, 158-8501, Japan

SOURCE: Journal of Pharmacological Sciences, (2004) Vol. 94, No. Supplement 1, pp. 36P. print.
Meeting Info.: 77th Annual Meeting of the Japanese Pharmacological Society. Osaka, Japan. March 08-10, 2004.
Japanese Pharmacological Society.
ISSN: 1347-8613 (ISSN print).

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 12 May 2004

Last Updated on STN: 12 May 2004

L5 ANSWER 19 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2003:555864 BIOSIS

DOCUMENT NUMBER: PREV200300551249

TITLE: ATP-induced three types of pain behaviors including allodynia.

AUTHOR(S): Inoue, Kazuhide [Reprint Author]; Tsuda, Makoto [Reprint Author]; Koizumi, Schuichi [Reprint Author]

CORPORATE SOURCE: Sect. Neuropharmacol., Div. Pharmacol., National Institute of Health Sciences, Tokyo, 158-8501, Japan

SOURCE: Drug Development Research, (August 2002) Vol. 56, No. 4, pp. 553. print.
Meeting Info.: Seventh International Symposium on Adenosine and Adenine Nucleotides. Queensland, Australia. May, 2002.
ISSN: 0272-4391 (ISSN print).

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 26 Nov 2003

Last Updated on STN: 26 Nov 2003

L5 ANSWER 20 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2003:223178 BIOSIS

DOCUMENT NUMBER: PREV200300223178

TITLE: Mechanism underlying UTP-evoked mechanical allodynia.

AUTHOR(S): Himaki, Daisuke [Reprint Author]; Koizumi, Schuichi; Mizokoshi, Akito [Reprint Author]; Tsuda, Makoto [Reprint Author]; Shigemoto-Mogami, Yukari [Reprint Author]; Inoue, Kazuhide [Reprint Author]

CORPORATE SOURCE: Div. Biosignal., Natl. Inst. Hlth. Sci., Tokyo, 158-8501, Japan

SOURCE: Journal of Pharmacological Sciences, (2003) Vol. 91, No.

Supplement I, pp. 201P. print.
Meeting Info.: 76th Annual Meeting of the Japanese
Pharmacological Society. Fukuoka, Japan. March 24-26, 2003.
Japanese Pharmacological Society.
ISSN: 1347-8613 (ISSN print).

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 May 2003
Last Updated on STN: 7 May 2003

L5 ANSWER 21 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:213125 BIOSIS
DOCUMENT NUMBER: PREV200300213125
TITLE: The expression and physiological function of P2Y6
receptor in rat microglial cells.
AUTHOR(S): Shigemoto-Mogami, Yukari [Reprint Author]; Koizumi,
Schuichi; **Tsuda, Makoto** [Reprint Author]; Sasaki,
Yo; Ohsawa, Keiko; Ishida, Seiichi; Kohsaka, Shinichi;
Inoue, Kazuhide [Reprint Author]
CORPORATE SOURCE: Div. Biosignal., Natl. Inst. Health. Sci., Tokyo, 158-8501,
Japan
SOURCE: Journal of Pharmacological Sciences, (2003) Vol. 91, No.
Supplement I, pp. 236P. print.
Meeting Info.: 76th Annual Meeting of the Japanese
Pharmacological Society. Fukuoka, Japan. March 24-26, 2003.
Japanese Pharmacological Society.
ISSN: 1347-8613 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 30 Apr 2003
Last Updated on STN: 30 Apr 2003

L5 ANSWER 22 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:290538 BIOSIS
DOCUMENT NUMBER: PREV200200290538
TITLE: Involvement of P2X **receptor** subtype in Ca²⁺
response evoked by low concentration of ATP in rat
microglia: Role in pain.
AUTHOR(S): **Tsuda, Makoto** [Reprint author]; Shigemoto-Mogami,
Yukari [Reprint author]; Mizokoshi, Akito [Reprint author];
Koizumi, Schuichi [Reprint author]; **Inoue, Kazuhide**
[Reprint author]
CORPORATE SOURCE: Sect. Neuropharmacol., Div. Pharmacol., National Institute
of Health Sciences, Tokyo, 158-8501, Japan
SOURCE: Japanese Journal of Pharmacology, (2002) Vol. 88, No.
Supplement 1, pp. 91P. print.
Meeting Info.: 75th Annual Meeting of the Japanese
Pharmacological Society. Kumamoto, Japan. March 13-15,
2002. Japanese Pharmacological Society.
CODEN: JJPAAZ. ISSN: 0021-5198.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 15 May 2002
Last Updated on STN: 15 May 2002

L5 ANSWER 23 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:267060 BIOSIS
DOCUMENT NUMBER: PREV200200267060
TITLE: Protective effect of cytokines against the death of PC12
cells caused by serum deprivation.

AUTHOR(S): Shigemoto-Mogami, Yukari [Reprint author]; Koizumi, Schuichi [Reprint author]; **Tsuda, Makoto** [Reprint author]; Obama, Tomoko [Reprint author]; **Inoue, Kazuhide** [Reprint author]
CORPORATE SOURCE: Div. Pharmacol., Natl. Inst. Hlth. Sci., Tokyo, 158-8501, Japan
SOURCE: Japanese Journal of Pharmacology, (2002) Vol. 88, No. Supplement 1, pp. 244P. print.
Meeting Info.: 75th Annual Meeting of the Japanese Pharmacological Society. Kumamoto, Japan. March 13-15, 2002. Japanese Pharmacological Society.
CODEN: JJPAAZ. ISSN: 0021-5198.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 May 2002
Last Updated on STN: 1 May 2002

L5 ANSWER 24 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:271878 BIOSIS
DOCUMENT NUMBER: PREV200100271878
TITLE: Characterization of Ca²⁺ signalings evoked by endogenous ATP in cultured rat hippocampal astrocytes.
AUTHOR(S): Koizumi, Schuichi; Shigemoto, Yukari; **Tsuda, Makoto**; **Inoue, Kazuhide**
SOURCE: Neuroscience Research Supplement, (2000) No. 24, pp. S105. print.
Meeting Info.: 23rd Annual Meeting of the Japan Neuroscience Society and the 10th Annual Meeting of the Japanese Neural Network Society. Yokohama, Japan. September 04-06, 2000. Japan Neuroscience Society; Japanese Neural Network Society.
ISSN: 0921-8696.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Jun 2001
Last Updated on STN: 19 Feb 2002

L5 ANSWER 25 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:215504 BIOSIS
DOCUMENT NUMBER: PREV200000215504
TITLE: Effect of P2 **receptor** antagonist PPADS in a rat model of surgical pain.
AUTHOR(S): **Tsuda, Makoto**; **Inoue, Kazuhide**
SOURCE: Japanese Journal of Pharmacology, (2000) Vol. 82, No. Suppl. 1, pp. 158P. print.
Meeting Info.: 73rd Annual Meeting of the Japanese Pharmacological Society. Yokohama, Japan. March 23-25, 2000.
CODEN: JJPAAZ. ISSN: 0021-5198.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 31 May 2000
Last Updated on STN: 5 Jan 2002

L5 ANSWER 26 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:282321 BIOSIS
DOCUMENT NUMBER: PREV199900282321
TITLE: Mechanisms of hyperalgesia induced by intrathecal administration of ATP analogue alpha, beta-methylene ATP in mice.

AUTHOR(S) : **Tsuda, Makoto** [Reprint author]; Ueno, Shinya
[Reprint author]; **Inoue, Kazuhide** [Reprint
author]
CORPORATE SOURCE: Division of Pharmacology, National Institute of Health
Sciences, 1-18-1 Kamiyoga, Setagaya-ku, Tokyo, 158-8501,
Japan
SOURCE: Japanese Journal of Pharmacology, (1999) Vol. 79, No.
SUPPL. 1, pp. 222P. print.
Meeting Info.: 72nd Annual Meeting of the Japanese
Pharmacological Society. Sapporo, Japan. March 22-25, 1999.
Japanese Pharmacological Society.
CODEN: JJPAAZ. ISSN: 0021-5198.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Jul 1999
Last Updated on STN: 28 Jul 1999

L5 ANSWER 27 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:282292 BIOSIS
DOCUMENT NUMBER: PREV199900282292
TITLE: Functional expression pattern of P2X receptors in rat
dorsal root ganglion neurons.
AUTHOR(S) : Ueno, Shinya; **Tsuda, Makoto** [Reprint author];
Katsuragi, Takeshi; Iwanaga, Toshihiko; **Inoue,**
Kazuhide [Reprint author]
CORPORATE SOURCE: Div. of Pharmacology, National Institute of Health
Sciences, Tokyo, 158-8501, Japan
SOURCE: Japanese Journal of Pharmacology, (1999) Vol. 79, No.
SUPPL. 1, pp. 110P. print.
Meeting Info.: 72nd Annual Meeting of the Japanese
Pharmacological Society. Sapporo, Japan. March 22-25, 1999.
Japanese Pharmacological Society.
CODEN: JJPAAZ. ISSN: 0021-5198.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Jul 1999
Last Updated on STN: 28 Jul 1999

L5 ANSWER 28 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:282276 BIOSIS
DOCUMENT NUMBER: PREV199900282276
TITLE: Effects of ATP on spontaneous synaptic currents through
glutamate or GABAA **receptor**/channels in rat
cultured hippocampal neurons.
AUTHOR(S) : **Inoue, Kazuhide** [Reprint author]; Ueno, Shinya;
Tsuda, Makoto [Reprint author]
CORPORATE SOURCE: Division of Pharmacology, National Institute of Health
Sciences, Fukuoka Univ., Fukuoka, Japan
SOURCE: Japanese Journal of Pharmacology, (1999) Vol. 79, No.
SUPPL. 1, pp. 46P. print.
Meeting Info.: 72nd Annual Meeting of the Japanese
Pharmacological Society. Sapporo, Japan. March 22-25, 1999.
Japanese Pharmacological Society.
CODEN: JJPAAZ. ISSN: 0021-5198.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Jul 1999
Last Updated on STN: 28 Jul 1999